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A MONTHLY DEVOTED TO THE
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JOHN FITCH LANDON, M.D., Editor

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POISONING AS THE CAUSE OF POLIOMYELITIS

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(Continued from the September issue)

PARALYSIS FROM MIASM *(continued)*

Pascalis[127] (1824) speaks about miasm as a cause of intermittent fevers. He says: "That such exposure proves generally unhealthful, it is readily granted, and they cause rheumatism, palsies, cramp, and a variety of other complaints."

Conventry[128] (1824), writing on endemic fever in the Genesee or Lake Country of New York State, says that it begins with the most violent arterial action; yet ends in the most marked nervous debility. He states that Hippocrates, many centuries ago, described the disease under the appelation "causus."

"During the years mentioned (1792-1795)," he says, "I can truly say that few days occurred, from the middle or latter part of July to the setting in of the frost, about the middle of October, that did not afford me the opportunity of visiting a patient with endemic fever. Having for forty years observed the species of fever spoken of, always accompanied by, and never independent of vegetable and animal decomposition, that it was dependent on, or connected with that process. The deleterious effects of vegetable and animal decomposition on the human body are established by innumerable facts with which the records of medicine abound. . . . Quarantines have been faithfully tried but are found wanting," he says.

Willoughby,[129] in a letter to Dr. Conventry in 1824, describing endemic fever in Herkimer County, New York State, says: "There was one symptom, and which might be considered as rather premonitory than otherwise, usually present this year (1803) which I have repeatedly noticed and subsequently, and which I do not recollect of seeing noticed by any writer. It was a certain diagnosis of fever, and by it I have been enabled to predict with certainty of fever before the patient was in the least aware of its danger, or hardly considered himself indisposed. This was a stiffness and aching sensation of the muscles in the back part of the neck. . . . I am certain that the decomposition of dead organized

matter, whether it be animal or vegetable, or both combined, when acted upon by heat and moisture, produces a poison (miasm if you please) which is the common exciting cause of itself over the whole system, deranging its functions."

Coventry[130] (1825) points out that the parts of the body most frequently affected in endemic fever are the head and the stomach. He speaks about perishable materials in basins, slips, docks, ponds, marshes and stagnant pools. "The cause is the same; and will be found accompanied by vegetable or vegetable and animal putrefaction. To produce and induce this process, heat and moisture are the principal agents; hence it prevails in those countries where these agents operate powerfully, and find pabulum to act on. The effects are varied by the idiosyncracy of the individual, and the intensity of the agent, as to induce respectable physicians to make distinctions where there is no difference. . . . The idea of contagion and importation is now so completely abandoned, that it would be an act of supererogation to dwell on the subject," Coventry says.

Fountain[131] (1827), writing on Intermittent Fever, points out that at no period within the space of fifteen years has intermittent fever prevailed so generally throughout the State of New York as during the last two years. "There is but one set of exciting causes, and this included all miasmic exhalations from marshes, soils, etc. The cause of intermittent fever is confessedly an invisible substance, the product of the play of chemical affinities among the materials of marshes, soils, etc., and all agents acting primarily on the nervous system; it follows of course, that the first evidence of disorder are manifestations of this tissue, acting under the combined influence of the morbific agent, and the inherent tendency of the living power to maintain its own movement. This complicated and confused respondence of the nervous system continues longer or shorter according to the feebleness of the morbific agent and the constitution of the living fiber. . . . I have known it induced by fright and by falling from a horse, and in these cases it came on instantly and pursued an uninterrupted course with its usual type. Besides it has been known to be a local disease affecting one arm only."

Fountain records the case of Eli Ganong of Putnam County, New York, who had a mild remittent fever. On the sixth day

he found that he could not move his hands or feet; and examination proved that they were paralyzed. The paralysis gradually progressed up his legs and arms, and on the fourteenth day they reached the muscles of respiration, and death ensued from their insubordination to the will. Sensibility of his mental faculties were unaffected.

Fountain points out how the various manifestations that he observed could result from arsenic but are frequently the effects of intermittent fever. "Yet every instance of the kind," he says, "occurring under the influence of arsenic would be attributed to this article." He speaks of a case of remittent fever which was denominated typhus fever, which he points out was a very convenient subterfuge for indolent practitioners.

Seymour[132] (1828, 1839) endeavored to show that poisons received into the circulation from the atmosphere act as mineral and vegetable poisons do on definite structures of the body. He points out that epidemic fever, according to the generally received opinion, originates from a poison arising from the marshes. This view, he says, has occupied the attention of physicians and men of observation from the earliest ages. "A correlation with the other poisonings has been pointed out for over a half century," he states, "It is by no means necessary, nor is it probable that miasm generated in the manner related should produce the same poison, or consequently affect the same texture uniformly; and thus we find epidemic fevers in which the poison appears to act directly on the brain, and various effusions in, or lesions of this viscus, are discovered after death."

Chambers[133] (1828) points out that remittent fever prevails in India and China, and other hot climates, in the unhealthiest season, viz., in August and September. The remittent fevers of England, he says, although differing from that of the hot climates, are the same disease in essence. This important observation appears to correlate the observations of Boerhaave[134] (1761), Shaw[135] (1823), and Goodeve[136] (1879) who pointed out that illnesses exist in India and China that result in paralysis.

Chambers emphasizes that this fever is not contagious; that physicians do not contract it from patients; and that other attendants do not contract it. He points out that there is no record of the disease spreading to other patients in a hospital.

Hawkins[137] (1829), England, speaks of remittent fever sometimes assuming an intermittent form, called arachnitis. Arachnitis of the base of the brain, he points out, produces rigidity of the muscles of the neck, whereas spinal arachnitis causes a general stiffness and curvature of the whole spine with locomotor powers affected. "It has happened," he says, "that the symptoms of other disorders which have been complicated with arachnitis have become the most prominent and have attracted the chief attention."

Mac Culloch[138] (1830) states: "Paralytic affections make their appearance also as a substitute for, or as a symptom in, both acute and chronic intermittents. Sometimes they occur in the course of it, and sometimes the original disease terminates in them."

Turner[139] (1832) describes the dry belly-ache of the West Indies, which he points out is of common occurrence there, proving fatal in many instances, and in others followed by paralysis. He felt that it was caused by lead poisoning. Musgrave[140] (1833), in a reply to Dr. Turner, denies that this disease is the result of lead poisoning. He felt that it was of malarious origin and that he had occasionally seen it in epidemic proportions. He points out that the disease occasionally occurs in town but is regarded as belonging to the country.

The modern virus theory, employed to explain the spread of poliomyelitis today, is reminiscent of animalcular theory employed to explain the miasmic diseases of the past. Caldwell[141] (1831) speaks of advocates of this theory as follows: "In their opinion putrefaction of marshes produced myriads of animalculae too minute to be detected by our senses or to become cognizable in any way, except by their effects on larger forms of living matter. These monads of life, as small and as numerous as particles of air, make their way into the human body by the pores of the skin, or in some other manner, mingle with the fluids, pierce and poison the solids, and spread corruption through the whole, then propagating their like with powerful fecundity, they issue from the bodies of the sick to invade those of the well, and thus the disease is rendered contagious. This hypothesis of animalcular contagion, however wild and irrational it may be deemed, has its advocates even now." Caldwell points out that the work of Lancisi, in 1717, was instrumental in discarding the animalcular

theory. Thus a "virus" theory became obsolete 233 years ago which was employed before then by some to explain illnesses that frequently resulted in paralysis.

Whereas the remittent and intermittent fevers of adults were generally attributed to miasm, those of children began to be associated by early writers with other factors, such as food, teething, foul bowels, worms, etc., as well as miasm.

Ashburner[12] (1834) says of infantile remittent fever: "That the train of events constituting the infantile remittent or developmental fever is but a part of that which leads to hydrocephalus acutus, that the one disease is a lower grade of the other is a proposition which seems to my mind too clear to require additional illustration."

Ramsbottom[13] (1835), England, points out that infantile remittent fever is most commonly occasioned in the older child by his being indulged in improper or unusual articles of diet with irritation of the teeth as an exciting cause.

Michael Underwood,[14] England, is considered to be the first to describe the disease that is now called Infantile Paralysis or Poliomyelitis. This supposition is based on the description of a disease entity which appears in the 2nd Edition of his book on diseases of children published in 1789. However, in the 9th American Edition of this same book, published in 1841, two years after Heine's reports[15] of Infantile Paralysis, this same disease would appear to have been relatively frequent in those days since under the heading of Paralysis or Palsy, for example, it is stated: "Paralysis is a more common disorder in infants and young children than writers seem to have imagined; it is confined to no age and attacks children in very different degrees, in the manner it does adult persons. It accordingly, sometimes, seizes the upper, and sometimes the lower extremities; in some cases, it takes away entire use of the limbs it has attacked, and in others, only weakens them. . . . Besides teething infants, whom it has said to attack, I have seen it in others who are older, and the finest of children, and it is often generally attended with a foul state of the bowels."

Armstrong's description of remittent fever appears to be pertinent to these cases of paralysis. He says: "The infantile fever, the infantile remittent, the biliary remittent of infants, or as it is sometimes, but improperly, called, the worm fever, generally

arises from indigestion, occasioned by food of improper quality, or too abundant in quantity. . . . It is a disease so far 'void of danger', that it frequently excites the greatest apprehensions, and sometimes proves fatal, or lays the foundation for other disorders, that are ultimately to end fatally."

Boling[146] (1846), writing on remittent fever says: "Heat, moisture, and the decay and decomposition of rich and luxuriant vegetation are the three principal causes to which its production is attributed, and certainly it gives manifestations of its presence under the existence of this combination more frequently than under any other." He speaks of a host of painful and anomalous neurological affections resulting from these agencies. "Remittent fever," he says, "has been divided into many varieties, dependent frequently upon the fancy of the writer, and having no real separate existence in nature: the different varieties passing by imperceptible shades into each other. The predisposition having already been engendered by the poisonous agent, there is no symptom more strikingly characteristic of remittent fever than excessive irritability of the stomach." He speaks about the bowels often being costive.

At about this time, Emerson[147] (1848) reported on the vital statistics of Philadelphia for the decennial period from 1830 to 1840. He points out that this city, with a population of 226,693, had a formidable number of deaths from diseases of the nervous system. "Convulsions claim, as usual," he says, "the largest proportion of victims recorded under a separate head, the whole amount during the decennial period being 2,985. Hydrocephalus is charged with 1,854 deaths, cephalitis 1,095, delirium tremens 826, apoplexy 704, palsy 471, epilepsy 108, insanity 104, tetanus 51, and chorea 10.

Stone[148] (1846), writing on miasmic diseases in the Western United States, asks and answers, "What then is a physician to treat, when called to the bedside of a sick patient? He has to treat a case of poison?"

Lawton[149] (1848) states that there is formed by decomposition a something which we call malaria, long known by its effect, being followed by a train of diseases peculiar to themselves, and known to be produced by no other morbific agent.

Kennedy[150] (1850), writing on the paralysis in early life, alludes

to those forms of the disease which occur subsequent to the fevers. "No matter what type they may assume," he says, "though it is most probably most usual to meet them after gastric and infantile remittent fevers, such cases are of tolerably frequent occurrence, and may be almost set down as forming a distinct group of themselves."

Churchill[151] (1850), in his book on diseases of infants and children, describes infantile remittent fever as follows: "It is a species of fever to which children of one year old to ten or twelve are very liable, characterized by one or more remissions, by pain of the belly, and sometimes, also, of the head, and by an unusual state of the alvine discharges. Such attacks are always attributed to worms by the older writers. Dr. Joy noted tenderness and intolerance of pressure in the upper part of the spine."

Churchill points out that there is irritation of the cerebro-spinal system in consequence of continued irritation of the intestines. Cold and teething are two principal causes of remittent fever as well as irregular or unwholesome food and it may occur in epidemics, he says. Sims, he points out, has described epidemics of infantile remittent fever which occur at the same time with the prevalence of a low nervous fever among adults. "The great difficulty in the diagnosis of remittent fever," Churchill says, "is from meningitis; and, no doubt, it is often a very difficult point."

Sawyer[152] (1858), writing "On the Vital Statistics and Causes of Mortality in San Francisco," states: "Cases of paralysis of every degree are of common occurrence here. In some instances it apparently has miasmic origin."

That infantile paralysis was of frequent occurrence at that time, and before, is indicated by Taylor[153] (1866) in his book on Infantile Paralysis, when he says: "There is sufficient reason for the fact that paralysis is of common occurrence in early life." The general practitioner apparently took these cases for granted and apparently designated them by various names. As a clinical entity infantile paralysis originated from specialists, rather than from practitioners, as the early reports of this disease clearly establish. The meagreness of the literature on the subject of infantile paralysis is thus explained by Taylor, an orthopedic specialist of New York Orthopedic Institute. "Only when some enthusiastic 'specialist' calls them from their hiding place—few in a given locality

but many in community—is there sufficient opportunity for that consecutive and multiplied observation necessary for establishing facts and making deductions." It is not surprising, therefore, that the early reports of infantile paralysis, as a disease entity and the end result of an illness not always ending in paralysis, should have originated from such orthopedic specialists as Underwood[154] (1789), Joerg[155] (1819), Stromeyer[156] (1833), Badham[157] (1834), Heine[158] (1838), and others; and later from neurologists after neurology also became a specialty. The increasingly frequent reports of operations for torticollis, strabismus, clubfoot, and curvature of the spine, as well as the establishment of orthopedic institutions in the first half of the 19th century, is of great significance.

Lanzi[159] (1876) made microscopic studies of the water of marshes, and as a result he considered the algae, passing into a state of putrefaction in the water, to be responsible for malarial fevers.

Lincoln[160] (1887), writing on, "The Climate and Health of Savannah" which covers a ten year period, states: "On the whole, the southern city presents a very high mortality from nervous diseases and miasmic fevers, and a rather high rate from digestive disorders."

Wood[161] (1889) states that a great many deaths, registered in South Australia during the period 1879-1880, were registered under the heading of paralysis. During these same years, he points out, malarial fever was very prevalent.

Bondurant[162] (1900) describes an epidemic of poliomyelitis in Green County, Alabama, in which there was much malaria at the same time. Included in this report are "some adult cases thought to be endemic of malarial neuritis." There was no evidence of poliomyelitis in the rest of the state.

The term "malarial fevers," which included a medley of diseases, was retained for some time after the discovery of the malarial parasite by Lavaran in 1880. Thus, it is to be noted that reports on the mortality in New York City during July 1907,[163] at a time when a severe epidemic of poliomyelitis was prevalent, recorded an augmented mortality from malarial fevers. There was also a rapid increase in the deaths from diarrheal diseases in infants and young children.

THE POLIOMYELITIS VIRUS: AN ENDOGENOUS PRODUCT

Since the claim of discovery of the so-called virus of poliomyelitis by Landsteiner[164] in 1908, every attempt made to show that this so-called virus enters the human body, under natural circumstances, to cause the disease has failed. It cannot be denied, of course, that a substance obtained from poliomyelitis victims, from sewage, flies, etc., commonly called a virus, exists and that it will produce a disease in experimental animals characterized by paralysis with lesions in the anterior horn cells of the spinal cord. Conclusive proof, however, that this so-called virus ever enters the human body, under natural circumstances, to cause the disease is conspicuously and definitely lacking.

There is a considerable amount of controversy concerning this portal of entry of the so-called virus into the human body. Thus, we find in the medical literature during the past decade such statements as the following: Aycock and Eaton[165] (1925): "Our present knowledge permits only speculation concerning the mode of infection"; Lumsden[166] (1938): "The views as to the portal of entry and communicability rest fundamentally and entirely on findings from experiments on monkeys. To some of us it does not appear to square with the facts obtained by epidemiological studies of the disease among human beings. It seems too elastic, too restful. On epidemiological grounds alone, it appears conceivable that poliomyelitis is not caused by a living microorganism or a virus, but by a toxin."; Neal[167] (1939): "The portal of entry of the virus is still a hotly debated question."; Sabin[168] (1939): "We possess no knowledge of the actual portal of entry into the human body"; McKhann[169] (1940): "The portal of entry of the virus has not been definitely established"; Smith et al.[170] (1945): "It is well-known that during and for some time after an epidemic the virus can be isolated from sewage, from outside toilets, occasionally from polluted and stagnant water, and in a few instances from flies which have been caught in nearby areas. Under some of the conditions the virus may remain active for several months. Theoretically, such non-human reservoirs would be expected to play an important role in the initiation of an epidemic and in the spread of the disease from person to person. Yet, in spite of the comparative ease with which polio may be experimentally transferred to monkeys from these reservoirs, it

has not been established that the virus in such form gives rise to the human disease. The possibility exists, but direct proof is still lacking." A perusal of contemporary reports on poliomyelitis (1950) shows similar statements, controversies, and speculations.

Although the literature for 35 years showed no record of "infection" of laboratory workers by the so-called virus of poliomyelitis, the accidental inoculation of an occasional laboratory worker during recent years has given renewed impetus to the hypothesis that a virus enters the human body to cause poliomyelitis. However, one could naturally expect if the so-called virus of poliomyelitis, whatever its nature, is accidently or intentionally injected into a human that it would obviously cause a disease similar to that produced in experimental animals by inoculation It must be admited, however, that this is an abnormal manner of acquiring a disease.

Experimental evidence, on the other hand, appears conclusively to have shown that the so-called virus of poliomyelitis does not enter the human body, under natural circumstances, to cause the disease. Experiments, intended to show that a virus enters the human body in a natural manner through the respiratory and gastro-intestinal tracts and the skin have been uniformly unsuccessful.

The experiments of Schultz and Gebhardt[171, 172] (1937) can be considered as almost conclusive proof that the so-called virus of poliomyelitis does not enter the human body by way of the respiratory tract. These workers found that the application of zinc sulfate as an astringent barrier when applied to the nasal mucosa of experimental animals prevented the passage of the so-called virus in the body. As a result of these experiments, zinc sulfate was applied to the nostrils of thousands of children in Toronto during an epidemic of poliomyelitis in 1937.[172] The results of these experiments, when analyzed, proved that the zinc sulfate applications had been ineffective in protecting children from poliomyelitis during that epidemic when compared with the results from the untreated control group.

A number of workers have likewise been unable to "infect" experimental animals by way of the gastro-intestinal tract. Howe and Bodian[174] (1939) had uniformly unsuccessful results in showing that the so-called virus enters the body at this point, as did

also Faber et al.[175, 176] (1943; 1948). These workers found that the gastro-intestinal mucosa in fact resisted the passage of the so-called virus of poliomyelitis. This fact was shown in their attempts to "infect" 67 monkeys by means of capsules heavily laden with the so-called virus. It was recently reported also that attempts to "infect" monkeys with the so-called Coxsacki virus by way of the gastro-intestinal tract were also uniformly unsuccessful.[177]

All of these unsuccessful attempts to "infect" animals indicate that although a substance, considered to be a virus, is found in the gastro-intestinal tract of cases of poliomyelitis, and subsequently in sewage, yet such evidence is in no way an indication that a similar substance ever entered the gastro-intestinal tract. One is forced to the conclusion that the gastro-intestinal tract contains, in cases of human poliomyelitis, and perhaps in the gastro-intestinal tract in the course of other diseases as well, a chemical substance which will produce a disease characterized by paralysis when *injected* into experimental animals.

Proof that a virus does not enter the human body through the skin, which it could almost certainly not do during epidemics of poliomyelitis except by an insect bite, is indicated by the fact that epidemics of this disease appear in many instances before the appearance of insects and continuance after insects have disappeared. The ineffectiveness of DDT and other insecticides in checking epidemics of poliomyelitis is further proof of the improbability of the skin as a portal of entry.

The portals of entry of a virus into the human body, under natural circumstances, to cause poliomyelitis has not only not been established, but the communicability of this disease after its supposed entry, has not been proven.

The theory that poliomyelitis is communicable has never been able to account for such anomalous and contradictory facts as the victimization of individuals who have had no contact with active cases; the non-communicability to doctors, nurses, and ward attendants; the absence of communicability to patients in hospitals and to individuals in communities when quarantines are not established; the rarity of multiple cases in the same family even where a child with poliomyelitis is known to have slept with another child; its greater incidence in small communities than in

large cities where crowding exists and where, consequently, poliomyelitis should extort a staggering number of victims; and the increase in epidemics of this disease in spite of improved hygiene and education regarding precautionary prophylaxis.

During the 1949 epidemic of poliomyelitis that swept the United States—the greatest epidemic of this disease recorded—contraction of it by definite contact with other cases was not established in an elaborate study by the New York State Department of Health. The United States Public Health Service in its studies also found the same negative answers during the same epidemic. Time Magazine,[178] commenting on these surveys, points out how, when and where people catch polio remained a mystery. Science News Letter[179] (August 19, 1950) indicates that quarantines for poliomyelitis are useless. It says: "Strict and heavy quarantines for infantile paralysis do not stop polio epidemics, health and poliomyelitis authorities agree. All attempts to stop polio by quarantine have failed and authorities now consider it foolish to enforce it."

Dissatisfaction with the virus explanation of cause of poliomyelitis appears to be evident from the fact that many theories, apposed to the virus theory, have been proposed since Landsteiner's discovery 42 years ago. Thus, there are found explanations of the cause of the disease in "globoid bodies," streptococci, poisons, allergy, vitamin deficiency, etc. Morse[180] (1929) in discussing the various etiological agents that have been described to account for this disease, i. e., filterable virus, hemolytic streptococcus, and globoid bodies, says: "They suggest the suspicion, however, that everybody may be wrong as to the etiological factor in infantile paralysis and that it may be something which has not even been thought of." This statement, made almost a quarter of a century ago, emphasizes the urgent need for unlimited research.

Some investigators have pointed out the fact that human poliomyelitis and the disease produced in experimental animals from human material, etc. are not the same disease. Toomey[181] (1935), for example, stated that intranasal and intracerebral inoculation of the poliomyelitis virus in monkeys does not produce the same disease that is seen in man.

The favorable results in the treatment of human poliomyelitis

by spinal drainage that have been noted by a number of workers[182-185] would appear to indicate: (1) benefit was derived by the draining off of a toxin, and (2) that human poliomyelitis and the experimental disease of animals are separate entities since spinal drainage in the case of the experimental animal has been found to be injurious.[186] In regard to the first of these premises, it is well-known that the poliomyelitis "virus" never has been recovered from the spinal fluid of either humans or experimental animals and that it would be impossible to drain off a virus that is fixed in the substance of the spinal cord. Therefore, the favorable results obtained in humans by spinal drainage and the unfavorable results obtained in experimental animals would appear to indicate that a poison is responsible for the human disease, whereas, a neurotropic enzyme resulting from this poisoning is responsible for the animal disease and it would be impossible to drain it off.

Toomey[191] (1939) insisted that the so-called poliomyelitis virus is an enzymatic catalytic agent, a fact that appears to reconcile the virus and the poison theories of this disease. Toomey found, for example, that the combination of bacterial or intestinal toxin and also peach skin filtrates accelerated the effect of the so-called virus when both were injected simultaneously into experimental animals.

Evidence that the human and experimental diseases are separate entities appears to be indicated by frequent observations that the heart is involved in cases of human poliomyelitis. Reporting on 35 cases of poliomyelitis in which necropsies were performed, Ludden and Edwards[192] (1949) suggest that the myocarditis in 14 cases was probably caused by the poliomyelitis virus. It was suggested that it might be possible to demonstrate the virus in such lesions and experimentally to produce them with the virus. This assumption is difficult to reconcile with the facts that the so-called virus of poliomyelitis has never been found in the blood and that it is neurotropic in action.

As further proof of the distinction between human poliomyelitis and the experimentally produced disease is the fact that drugs that have proved to be effective in the treatment of the latter have been ineffective in the former. The vaccines of the "virus" that have been prepared and proved to be effective in

controlling the experimentally produced poliomyelitis in animals, have caused in humans (as in the case of laboratory workers), the paralysis which the vaccine sought to prevent.[193]

A revision of our views regarding the virus concept of cause of poliomyelitis is called for on the basis of the fact that "viruses" can be produced in the body by poisons, and that inclusion bodies, which supposedly indicate products of virus activity or the elementary virus bodies themselves, can be found in poisoned animals. This work has been reviewed by Scobey.[194]

Although Landsteiner and others have been able to inject emulsions of spinal cord material into experimental animals to cause a paralytic disease, a number of workers have also caused paralysis in experimental animals simply by injecting normal nervous tissues.[195] This fact would indicate that some of our modern studies relating to the so-called virus of poliomyelitis require careful reappraisal. Lumsden[196] (1949) states: "Brain tissue from many mammalian species contains some non-infective chemical agent which is specifically pathogenic to the mammalian nervous system." He tested over 300 guinea pigs, 150 rats, and 12 rabbits and experimentally produced encephalomyelitis with normal brain material.

COMMENT

This report shows that there are many opportunities for poisoning with resultant anterior poliomyelitis during the seasons when epidemics of this disease are prevalent. The significant changes in fruits and vegetables, as a result of drought and lack of water; toxicity of food from bacterial contamination; chemical poisoning of fruits and vegetables during growth by fertilizers, other chemicals, and insect sprays; and chemical changes in water from decay of organic material by natural processes or bacterial decomposition with such organisms as pseudomonas or the botulinus organism have all been either ignored or not adequately correlated as factors in producing human poliomyelitis. Yet, this wide variety of poisons does produce essentially the same result, i. e., flaccid paralysis with changes in the anterior horn cells—poliomyelitis.

Putnam[197] (1888) stated, regarding poisons in general: "The very fact that such widely different agents as lead, arsenic, alco-

hol, sulphide of carbon, and the poison of the tubercular, and other bacteria induce on the whole such similar types of neuritis, suggests that there is a common factor at work which is independent of the particular agent, and must consist in some peculiarity of the affected tissues. If this is so, then a variation of the symptoms which is often met with in one of these forms of poisoning, such as that from lead, will be likely to occur to a greater or less extent in the rest, when the conditions are favorable. These peculiarities in the local accumulation of the poisonous agent must certainly affect the symptoms, even if it be true that the degree of vulnerability of the special tissues, and not the amount of the poison present, that chiefly determines whether or not paralysis will result."

The above statement indicates not only an individual susceptibility to poisons but likewise a secondary factor. It is highly possible that this secondary factor, common to most poisons, is a vitamin deficiency, presumably a multiple deficiency. This fact is suggested by the work of Hart et al.[66], Mellanby[70], and Reyes[71] which has been previously considered. There is also to be considered the sequelae of poisoning as exemplified in alcoholism with its resultant polyneuritis, paralysis, Korsikoff's syndrome and Wernike's encephalitis, for many years considered to be due to the specific effects of alcohol on the nervous system, but which are now generally recognized as caused by vitamin B_1 deficiency as secondary to alcoholism.

Post-diphtheritic and post-typhoid paralysis, formerly thought to be the direct effect of the specific bacterial toxins are now also regarded as due to a vitamin B_1 deficiency secondary to these toxins. The reports that have appeared in the literature of the development of poliomyelitis as a sequel to acute infectious diseases of childhood and secondary to pertussis inoculations are evidently of the same nature. The effectiveness of vitamin B_1 reported by some workers in the treatment of poliomyelitis would appear to indicate that a secondary vitamin B_1 deficiency does in fact exist in poliomyelitis.

One of the logical approaches in the investigation of the poliomyelitis problem would appear to be the search for a poison in the food and water of victims of this disease. Advice given in an editorial in the Boston Medical and Surgical Journal[78] over half

a century ago appears to be extremely important in emphasizing this point. It says: "One of the most significant advances of the past few years has undoubtedly been the attention paid to the importance of various toxic causes in the production of disease. The work of Van Giesen of New York and of many foreign investigators has forced upon us the conviction that the etiological problems of medicine may be much more intelligently met by widening our conception of the idea of poisons, and studying with every possible means at the disposal of our laboratories the chemical questions which such conception involves. *Unquestionably progress lies in this line of investigation.* It is self evident, as civilization advances and new products are used more and more in the arts and in our homes, that new dangers must necessarily arise, and a constantly growing list of so-called extrinsic poisons be brought into our daily life."

Truthfully, progress was being made in the investigation of poliomyelitis before the discovery of the so-called virus. Toxicologists and bacteriologists were working simultaneously on the problem, with limited funds, and apparently reporting important results. For example, it was reported from scientists in these branches of medicine that poliomyelitis could be produced both by organic and inorganic poisons as well as by bacterial toxins. However, these investigations lost support when the so-called virus theory came to be considered by some to be the full and final answer to the problem. Funds for poliomyelitis research were from then on designated only for virus studies. Another factor, and perhaps the most important one, that interrupted this pioneer work and that still stands as a barrier to unlimited research today, was the compulsory reporting of poliomyelitis as a communicable disease, which implies legally that this disease is caused by a germ or a virus.

Our forefathers, who were responsible for the fundamentals of medicine on which modern medical science is still based, incriminated food and water as the cause of diseases that not infrequently resulted in paralysis. The foundation of our knowledge of poliomyelitis was laid by observing men like Underwood, Badham, Heine, Colmer, West, Duchenene, Landry, Charcot, Erb, Gowers, Strümpel, Wickman, and many others who did not have access to the spinal puncture needle to assist them in their diagnosis

of this disease. The spinal puncture was not generally employed in the diagnosis of disease until several years after it was introduced by Quinke,[139] in 1891.

The fundamental principles of investigation of epidemics advocated by Hippocrates,[98] The Father of Medicine, included meteorology, astronomy, dietetics, physical and chemical examination of water, the effect of weather conditions on vegetation, etc. These potential factors are being ignored in the mandatory requirement of an exogenous virus as the cause of poliomyelitis. Hippocrates' statement regarding these fundamental principles of epidemic investigation: "*For from these things he must proceed to investigate everything else.*" appears to have been the mode of investigation of disease for centuries. Today under the pressure of the exclusive virus theory of cause of poliomyelitis the wisdom of Hippocrates is obscured. Dauer[1] (1938) makes the following significant statement: "The aim of epidemiology is to find out and expose, as far as humanly possible, why diseases are as they are, and to learn the rules under which they act." This statement implies, as advocated by Hippocrates over 2000 years ago, unlimited research and the taking of every possibility into consideration.

To repeat, the predominating factor that obstructs our progress in poliomyelitis investigation today and the continuance of the valuable observations made by the pioneers of medicine is obviously the "virus". This more or less general acceptance of an exogenous "virus" as the cause of poliomyelitis has obviously been influenced by subsidized exclusive "virus" research, mandatory public health laws, and continuous, widespread "virus" publicity. Poliomyelitis investigation obviously calls for a revision of exclusive concepts and for unlimited research.

SUMMARY

1. A number of theories, other than the virus theory, have been proposed to explain the epidemiology of poliomyelitis. The "poison theory", which for centuries from the time of Hippocrates has been emphasized to explain the paralytic diseases of the past and the poliomyelitis of more recent times, has been more or less generally ignored for almost half a century. Proof that poisons can cause poliomyelitis is given in this report.

2. The so-called virus of poliomyelitis is considered to be an endogenous chemical substance resulting from this poisoning. Human poliomyelitis and the experimental animal disease are regarded as separate disease entities.

3. It is pointed out that the more or less general acceptance of an exogenous virus as the cause of poliomyelitis has been influenced by exclusive virus research; mandatory public health laws that legally make poliomyelitis a communicable disease; and widespread virus publicity.

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RESULTS OF OPERATION IN 40 CASES OF CONGENITAL HEART DISEASE. (Thorax, London, 5: 73, March 1950). Wilson reports the results of follow-up on 40 of 63 patients operated on for congenital cyanotic heart disease at the University Hospital in Copenhagen. No case was included in which less than three months had elapsed since the operation, and only 6 were included in which there had not been at least six months' observation. Thirty-five of the 40 patients had tetralogy of Fallot; 2 patients had pulmonary stenosis with interatrial septal defect; 2 had an underdeveloped right ventricle, and 1 was thought to have aortic stenosis associated with an interatrial septal defect and a small functioning patent ductus arteriosus. Two of the 5 deaths that occurred in these 40 cases must be considered as anesthetic deaths. In assessing the results of the operation, the author attempted to view each case as a whole, with attention to improvement in color, height and weight increase, absence or sign of cardiac insufficiency as well as to the important feature of increased or unchanged exercise capacity. A final analysis revealed that the result was excellent in 8, very good in 10, good in 6, fair in 8, poor in 1 and unchanged in 2.—*Journal A.M.A.*